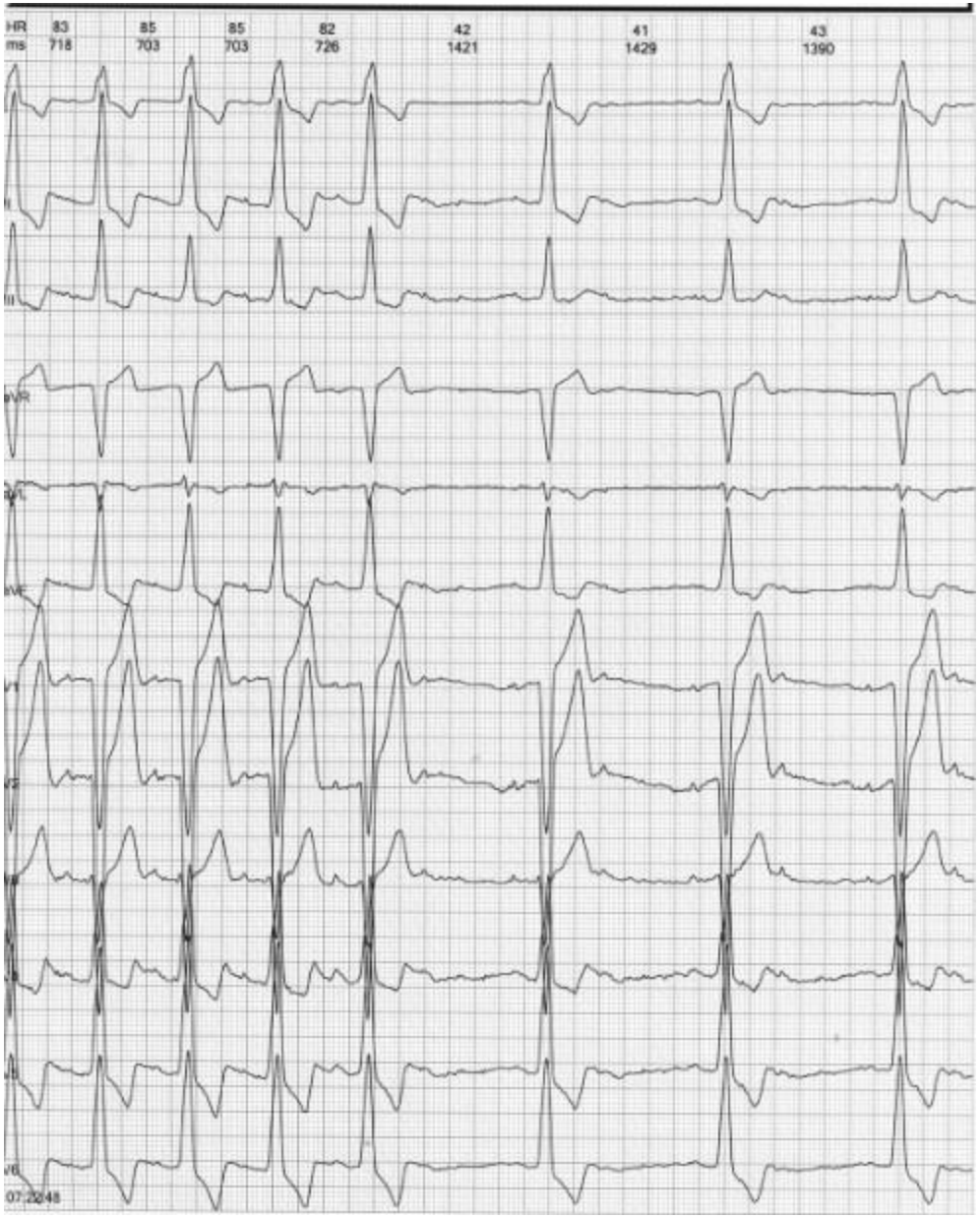
Senior man with syncope and dynamic intraventricular conduction defect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aVL
aVF
V1
V2
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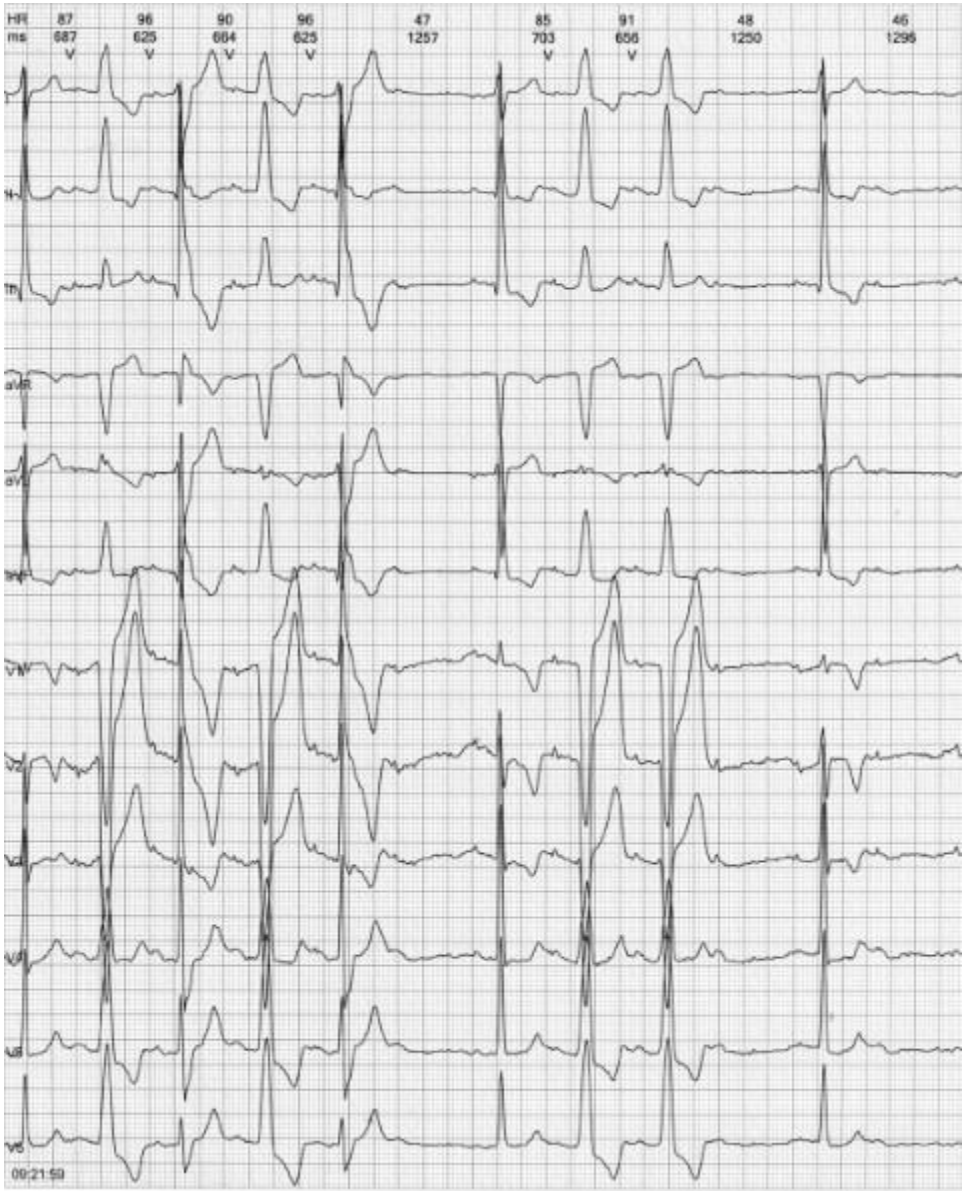
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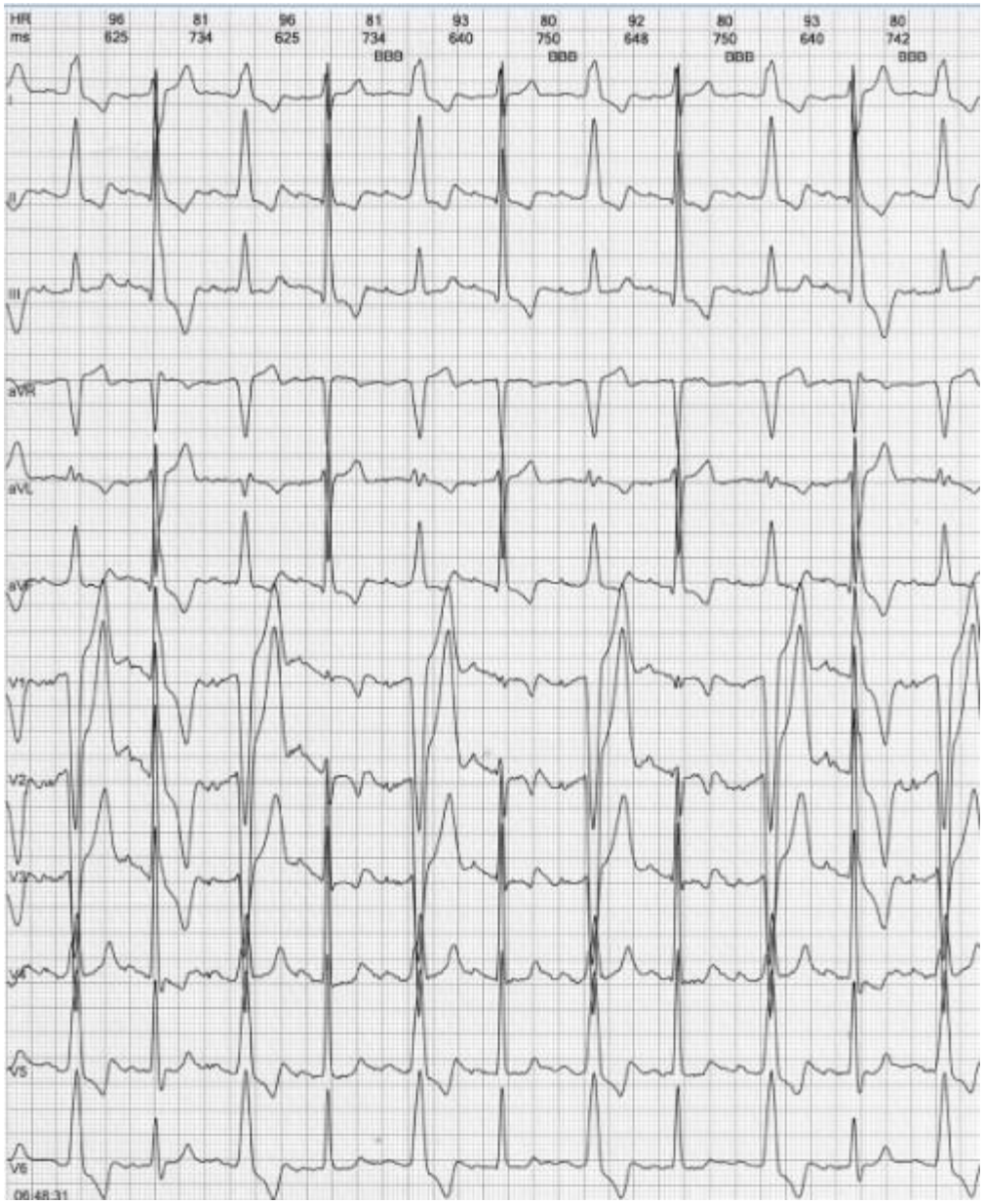
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V3
V4
V5
V6
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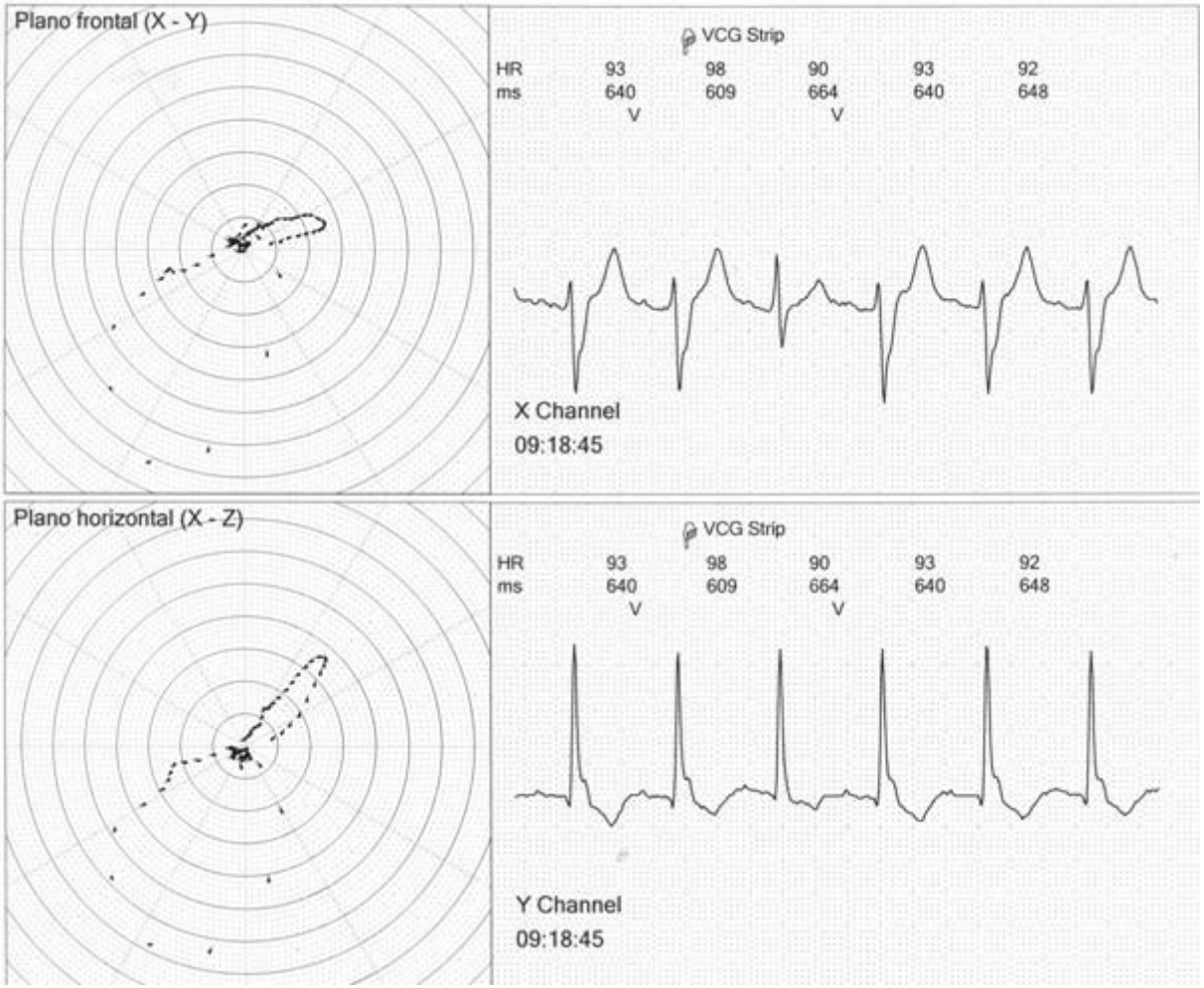
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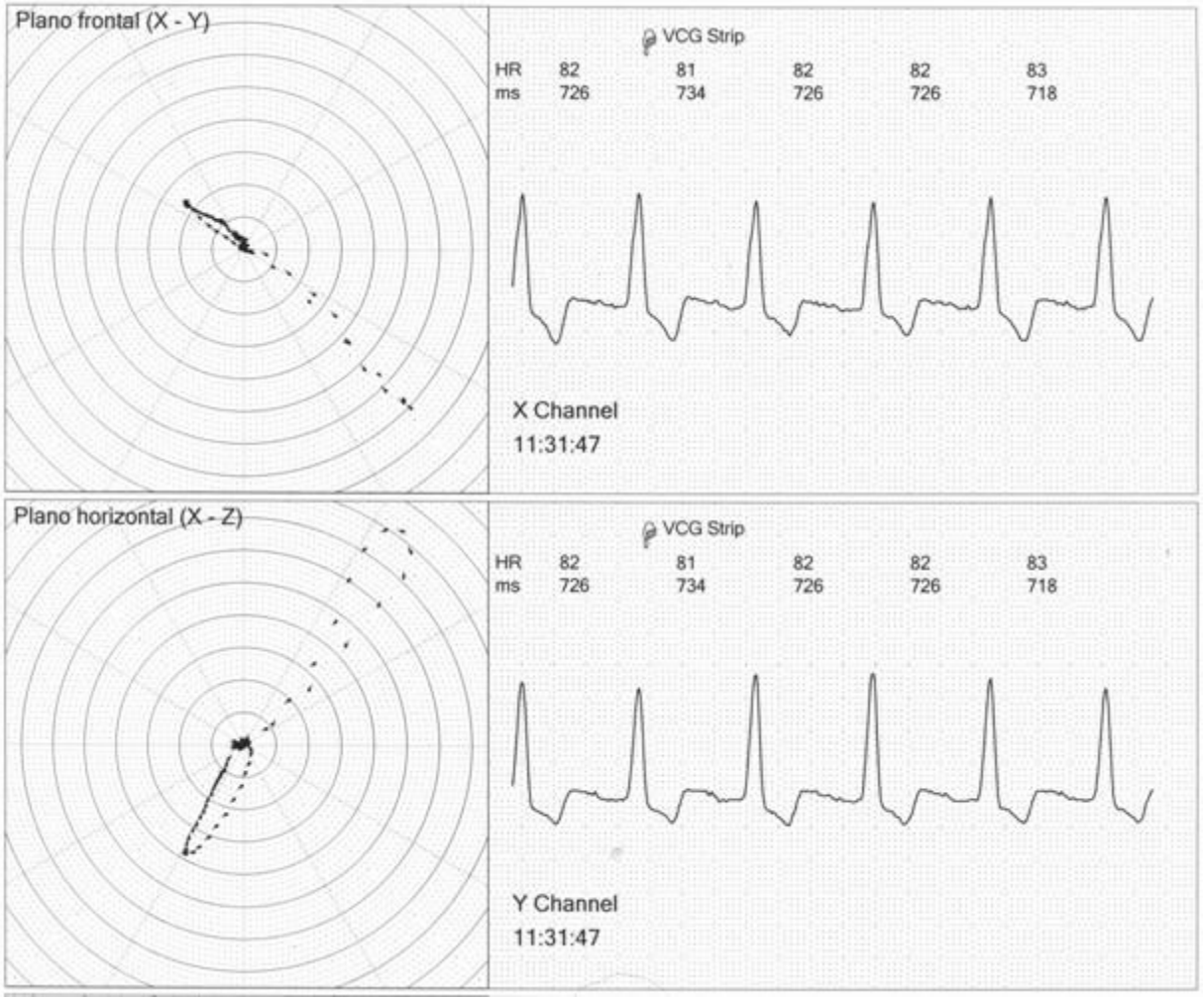
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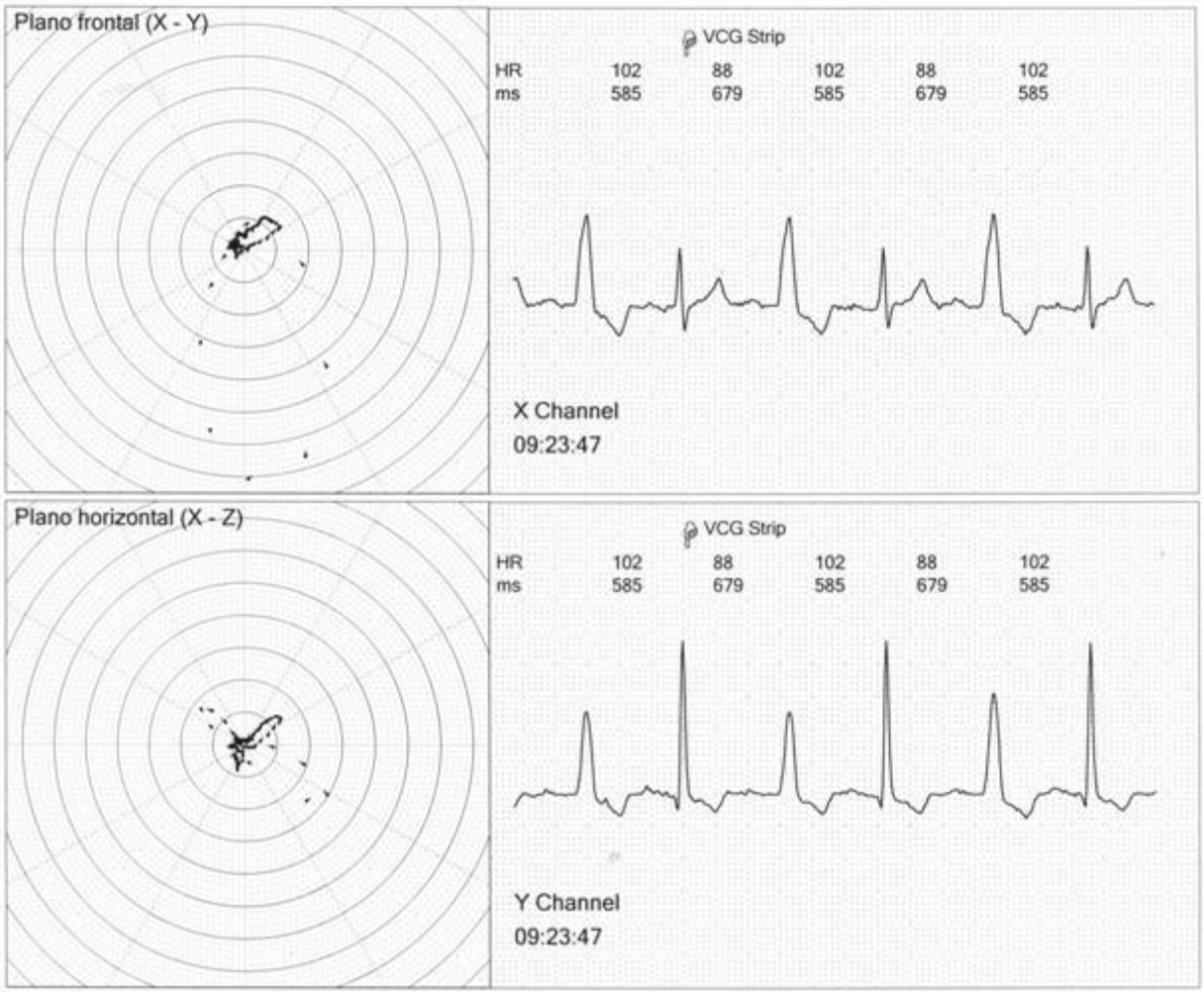
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Plano horizontal (X - Z)
VCG Strip
HR 102 88 102 88 102
ms 585 679 585 679 585
Y Channel
09:23:47

Colleague’s opinions

First tracing LBBB as well as 2:1 AV block with ventriculo phasic phenomena.2nd tracing shows alternate LBBB with RBBB and left posterior hemi block.Note the change in PR interval with change in BBB pattern as well as normally conducted beats with narrow QRS perhaps due to equal delay in bundles..The 3rd tracing shows the bilateral BBB pattern terminated by blocked P wave which introduces a pause followed by narrow conducted QRS.

The bilat BBB pattern associated with a relatively short PR as well as change in PR associated with change in bundle pattern all suggest infra nodal disease and need for pacing.

The “normal” also show minor RVCD

Melvin Scheinman

The ECG shows bilateral bundle branch block. Left bundle branch block alternating with right bundle branch block with left posterior hemiblock in one ECG. We also see Mobitz type II in another tracing.
Thus explains his syncope. He needs a pacemaker.

Very nice tracings!

Thank you,

Mario González

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http://www.pennstatehershey.org/heartandvascular

Spanish
Estimado Andrés: una vez más estás mandando figuras interesantes!!!!
Analizando sólo el ECG (el VCG no lo veo bien) me impresiona un bloqueo AV trifascicular paroxístico manifestado por: ondas P conducidas con BRI, otras con BRD más HBP, otras con HBP sólo y otras no conducidas. El hisiograma sería diagnóstico en las P no conducidas si se acompañan del haz de His. Dado que el diagnóstico del HBP es clínico-electrocardiográfico habría que descartar EPOC, patología cardíaca derecha o corazón vertical.
De tratarse de ésto, los síncopes no tienen pródromos, tampoco factores predisponentes y la recuperación de la conciencia es rápida y total. Me gustaría saber más de los episodios para seguir aprendiendo. Por último es el manejo: ¿ internación o ambulatorio? ¿¿¿EEF previo o no hace falta??
Saludos a todos
Daniel Dasso, M.D.
Buenos Aires

Spanish

Querido Andrés: el caso es sumamente interesante el único problema es que uno tiene muchos problemas en su interpretación por el cambio y ubicación de las fuerzas de los complejos QRS.

Comencemos con la onda P, parece sinusal con una duración de alrededor de 220 mseg con una frecuencia de aproximadamente 100 x min, con trastornos de conducción intra e interauricular importante si la pudiese seccionar y ampliar. Me impresiona por lo poco que puedo observar en el plano horizontal que primero se orienta con rotación horaria hacia el campo anteroinferior izquierdo luego al punto cero para continuar con un buccle que no puedo vislumbrar si es horaria o antihorario en el plano posterior izquierdo. Si esto es así la activación de ambas aurículas se produciría en forma céfalo caudal (normal) pero con un retrazo importante en la activación de la izquierda. Algunas P se bloquean produciendo un Bloqueo AV 2:1.

Cada complejo QRS está precedido por una onda P, el comienzo es muy rápido y los empastamientos están ubicados en las partes medias o finales por lo cual supongo que son conducidos. (Uno debe descartar que no tengamos un ritmo ventricular que se este fusionando)

Para los complejos QRS por un lado me gustaría ver el plano sagital y por el otro querría saber cuantos mseg existen entre las comas (no es ni 2, ni 2,5 mseg). Este hecho me dificulta saber cuanto mseg tardan en activarse las diferentes zonas miocárdicas.

Voy a empezar detallando lo que observo en el segundo VCG: BRI atipico. Las primeras fuerzas en el plano frontal se dirigen hacia arriba con rotación horaria y con un eje en los 45 ª con un bucle totalmente entrecruzado.

Estas primeras fuerzas podrían ser debidas a

- una necrosis inferior, pero el resto del complejo QRS se debería dirigir hacia arriba por lo cual la descarto.

-Agrandamiento del VD; las primeras fuerzas del bloqueo de RI deberían estar conservadas (tendría que agregarle otra patología para que esto fuese posible)

- bloqueo posteroinferior de la rama derecha. Este comportamiento lo he observado en e algunos latidos aberrantes en paciente normales. En este caso es la explicación que mejor encuadra por ser la mas simple.

Primer VCG; el primer vector nace a la derecha arriba y atrás (descartamos BRI). No puedo medir cuanto tiempo tarda en activarse el VD, por el problema de las comas. El vector espacial máximo (que lo supongo, porque no tengo el plano sagital para corroborarlo) no se a cuantos mseg aparece, pero se ubica en el cuadrante antero inferior derecho a los 120ª en el plano frontal. En ambos planos presenta rotación horaria. La duración del complejo QRS es de 160 mseg.

A todas mis especulaciones por la duración del complejo QRS le tengo que agregar un BRD

-Agrandamiento del VD + BRD?

-BRD+HPID?

Y por último el tercer VCG nos muestra un latido angosto con un eje en 90ª y rotación horaria en el plano frontal, con las últimas fuerzas dirigidas hacia el cuadrante posteroinferior del VD. No puedo argumentar que este complejo sea un ritmo de fusión entre los dos complejos QRS anteriores. Pero me inclinaría a pensar que puede existir un retraso en la conducción en las regiones postero inferiores del VD.

Esta es la explicación más sencilla, hay otras un poco más complicadas que de acuerdo a lo que sigan mostrando del caso se puede continuar especulando.

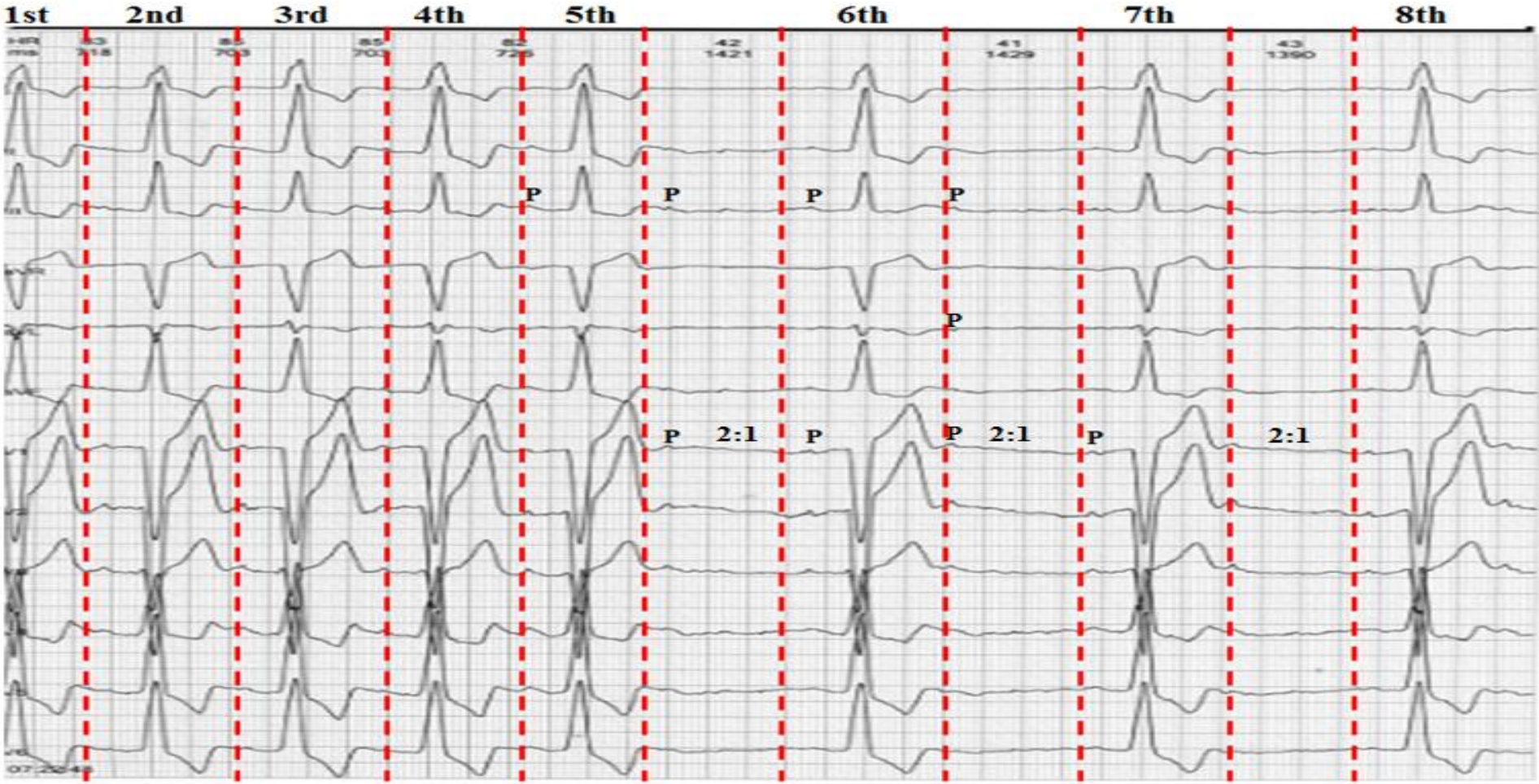
Muchas gracias Andrés por este caso tan bonito. Afectuosamente

Isabel Konopka

Final conclusions by

Andrés Ricardo Pérez-Riera
&
Raimundo Barbosa-Barros

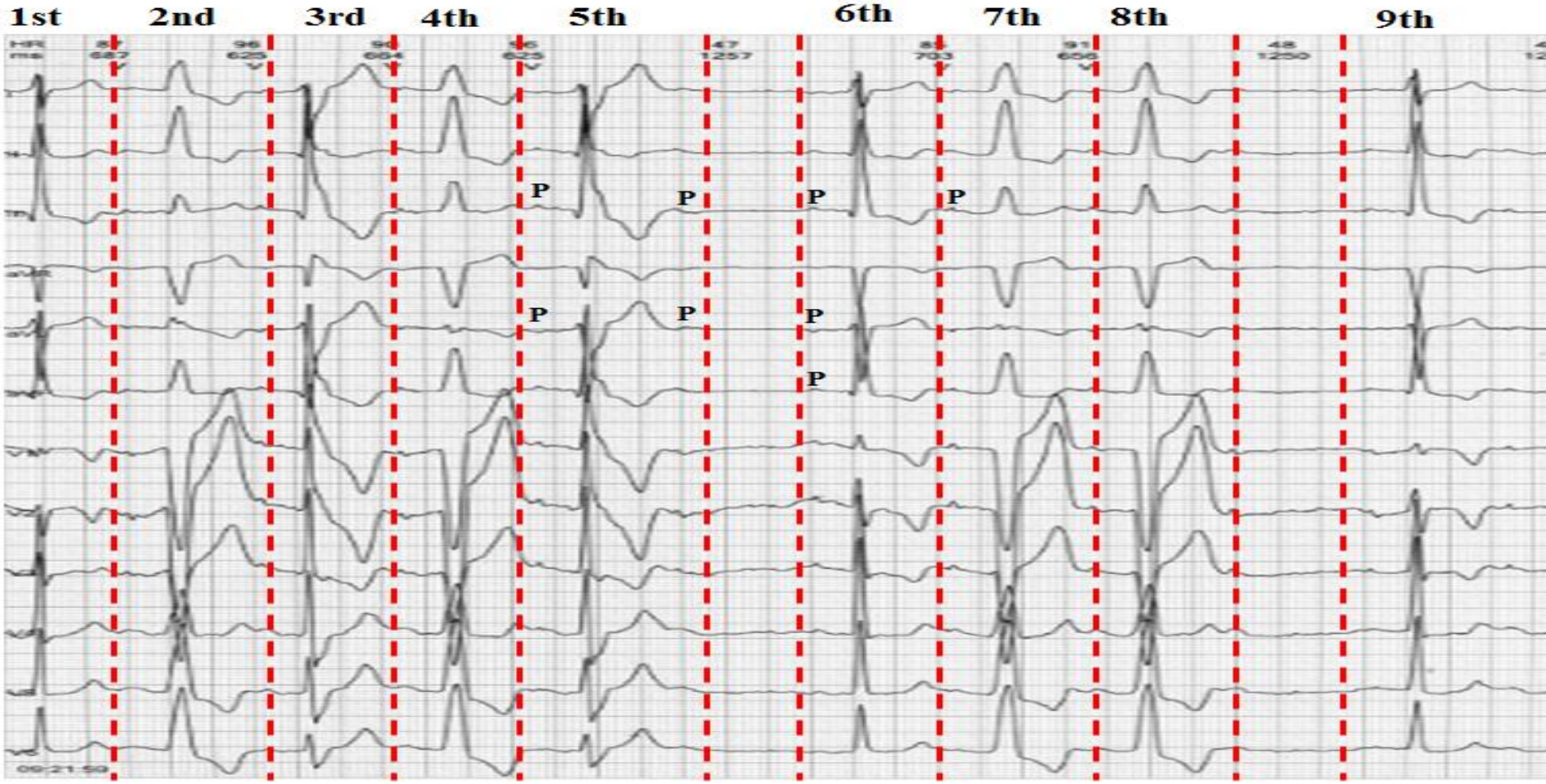
ECG 1



Todo o traçado apresenta padrão de bloqueio de ramo esquerdo com eixo elétrico do QRS em +60°. Os primeiros 5 batimentos apresentam P conduzidas com intervalos PR prolongados (240 ms: bloqueio AV de primeiro grau). As ondas P depois do quinto, sexto e sétimo batimentos são bloqueadas, configurando um bloqueio AV de segundo grau Mobitz II 2:1.

The trace shows LBBB pattern with QRS axis at +60°. The first 5 beats have conducted P-waves with prolonged PR intervals (240 ms: first degree AV block). The P waves after the fifth, sixth and seventh beats are suddenly blocked, indicating 2:1 second-degree AV block Mobitz II.

ECG 2

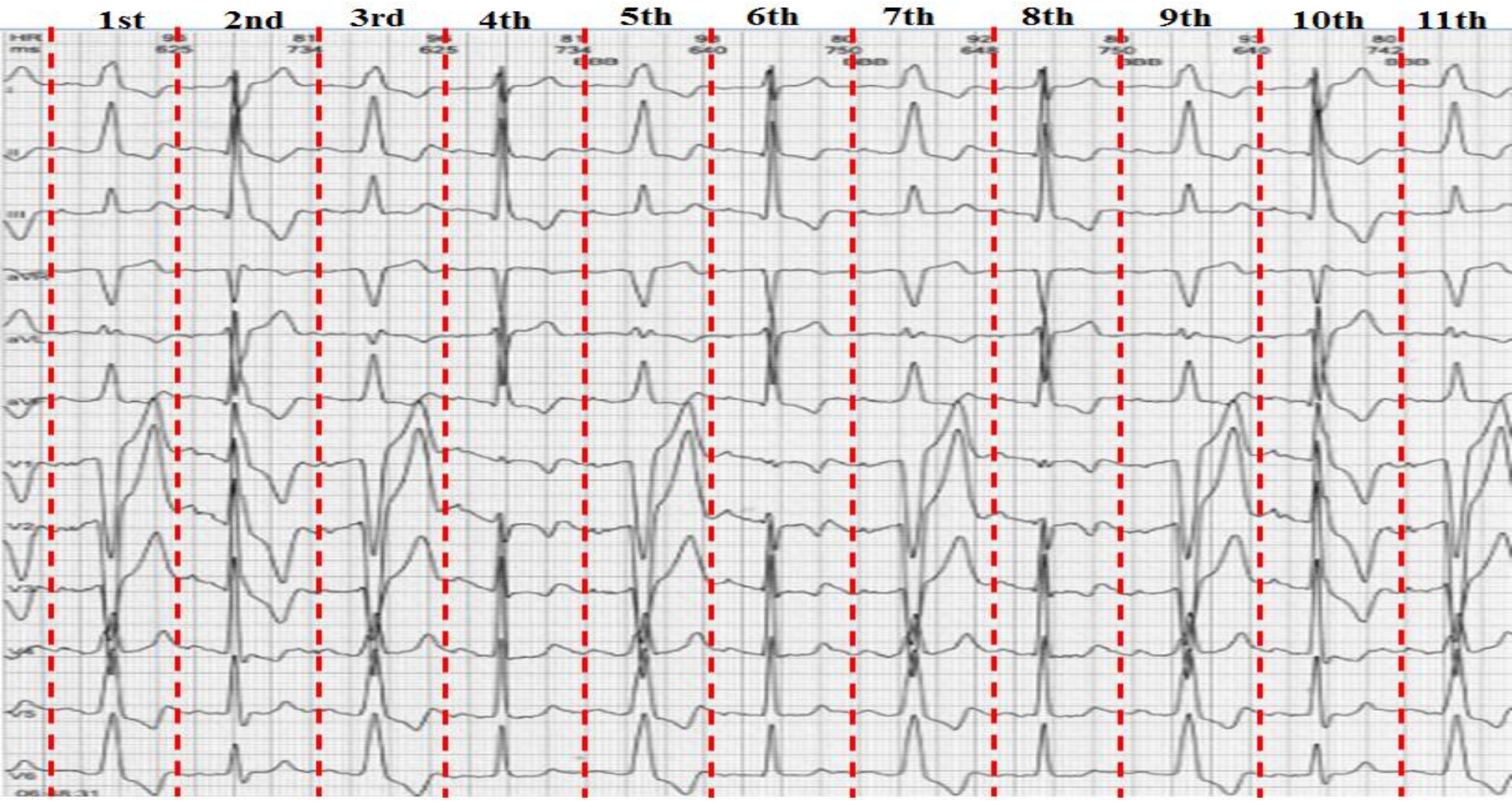


O segundo, quarto, sétimo e oitavo batimentos têm padrão de BRE. O terceiro e quinto batimentos têm padrão de bloqueio divisional ou fascicular póstero-inferior esquerdo (BDPIE), associado a BRD. O primeiro, sexto e nono batimentos possuem um grau menor de BDPIE. As ondas P após o quinto e oitavo batimentos estão bloqueadas, configurando um bloqueio AV de segundo grau 2:1 Mobitz II.

The second, fourth, seventh and eighth beats have LBBB pattern. The third and fifth beats have LPFB pattern associated with RBBB. The first, sixth and ninth beats show a lesser degree of LPFB.

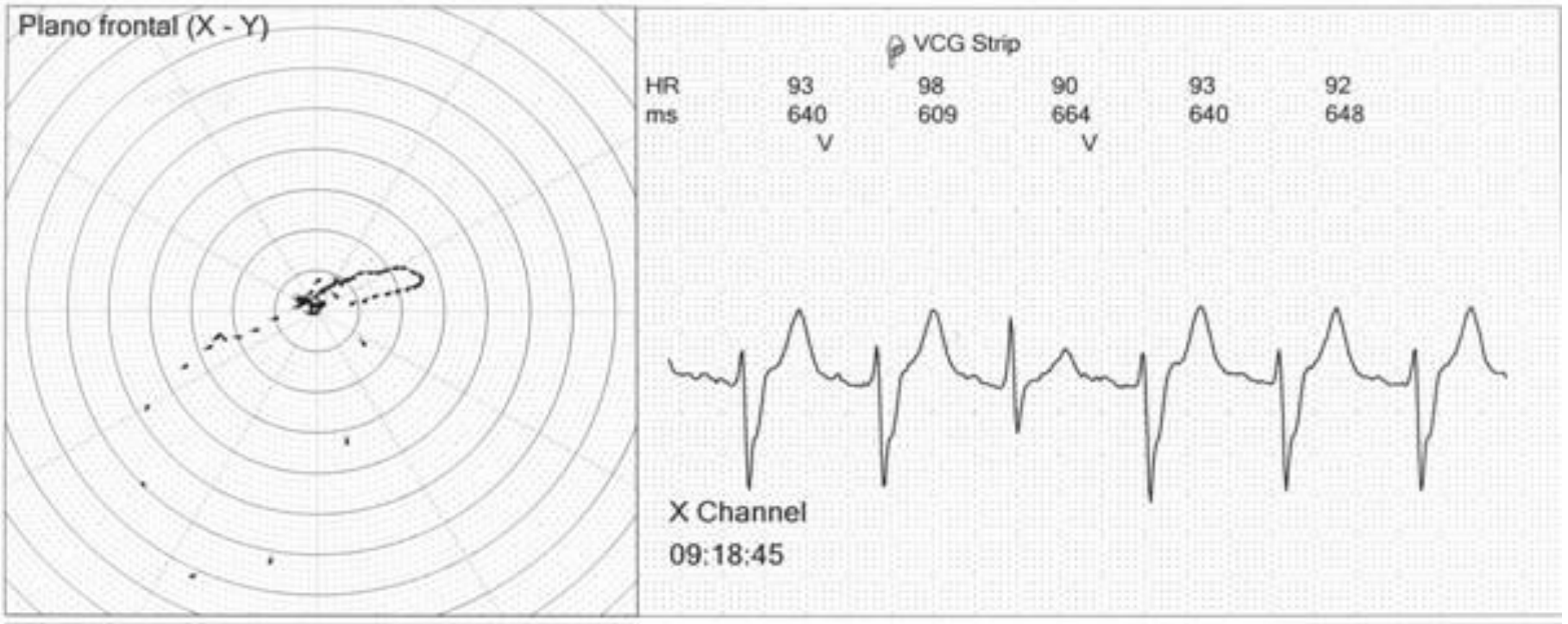
The P waves after the fifth and eighth beats are blocked, indicating a second-degree AV block 2: 1 Mobitz II.

ECG 3



O primeiro, terceiro, quinto, sétimo, nono e décimo primeiro batimentos têm padrão de BRE. O segundo e o décimo batimentos têm padrão de BDPIE + BRD. O quarto, sexto e oitavo batimentos têm um grau menor de BDPIE. Os intervalos PR estão prolongados (240 ms: bloqueio AV de primeiro grau).

The first, third, fifth, seventh, ninth and eleventh beats have LBBB pattern. The second and tenth beats have LPFB + RBBB pattern. The fourth, sixth and eighth rate have a lesser degree of LPFB. The PR intervals are prolonged (240 ms: first degree AV block).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VCG Strip
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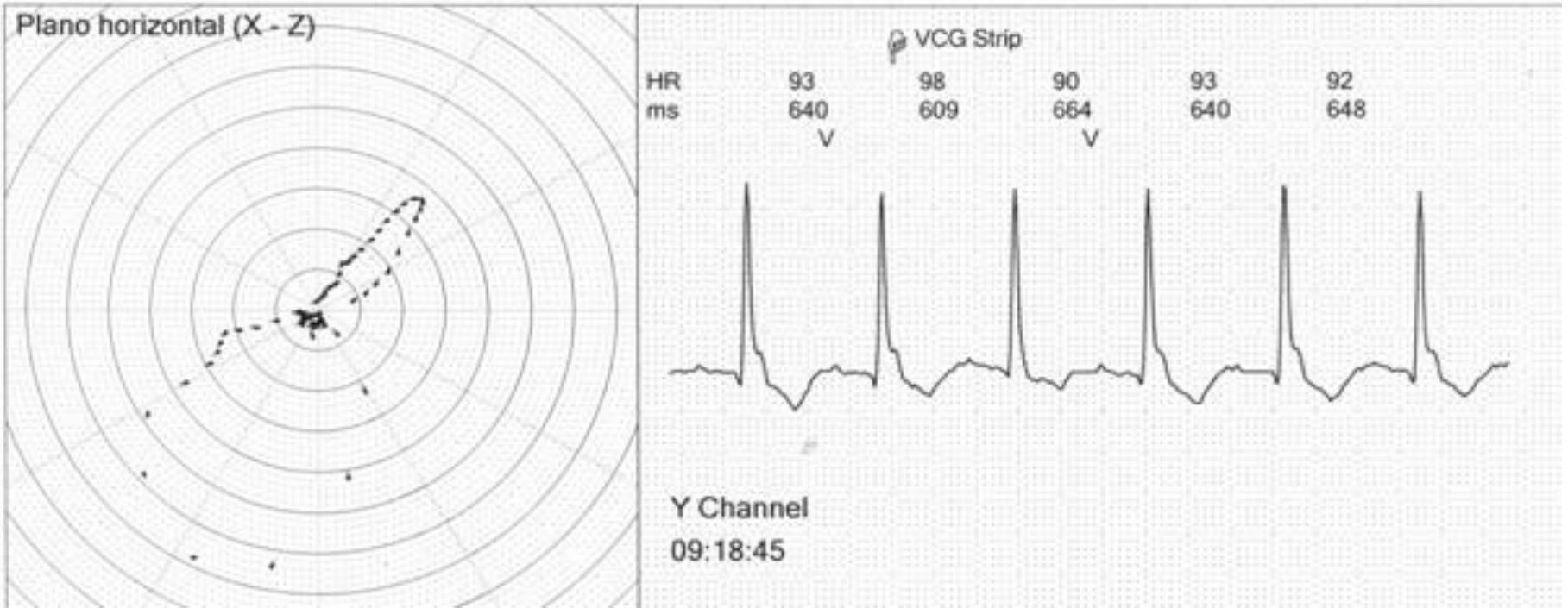
ms 640 609 664 640 648
V V
X Channel
09:18:45


Plano horizontal (X - Z)
VCG Strip
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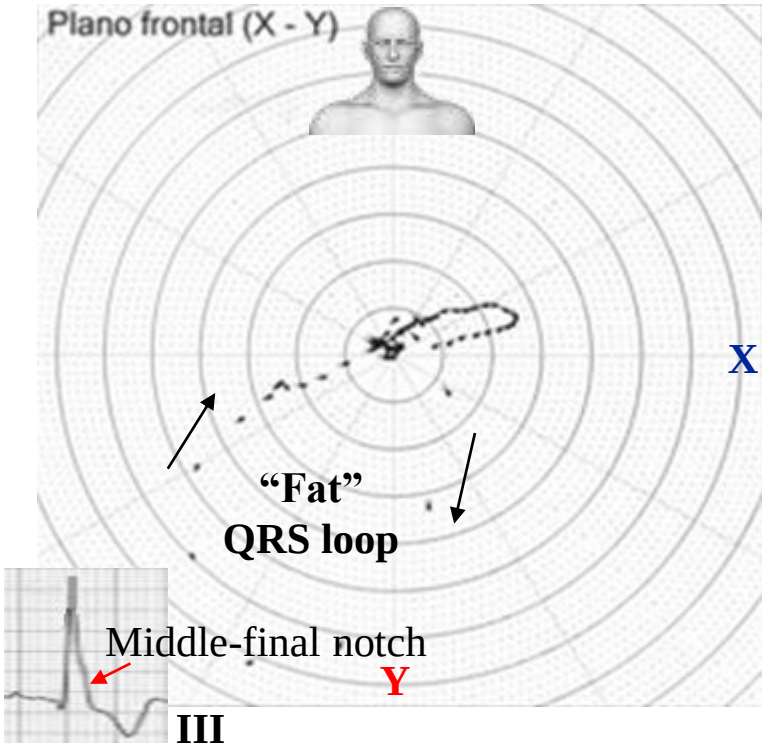
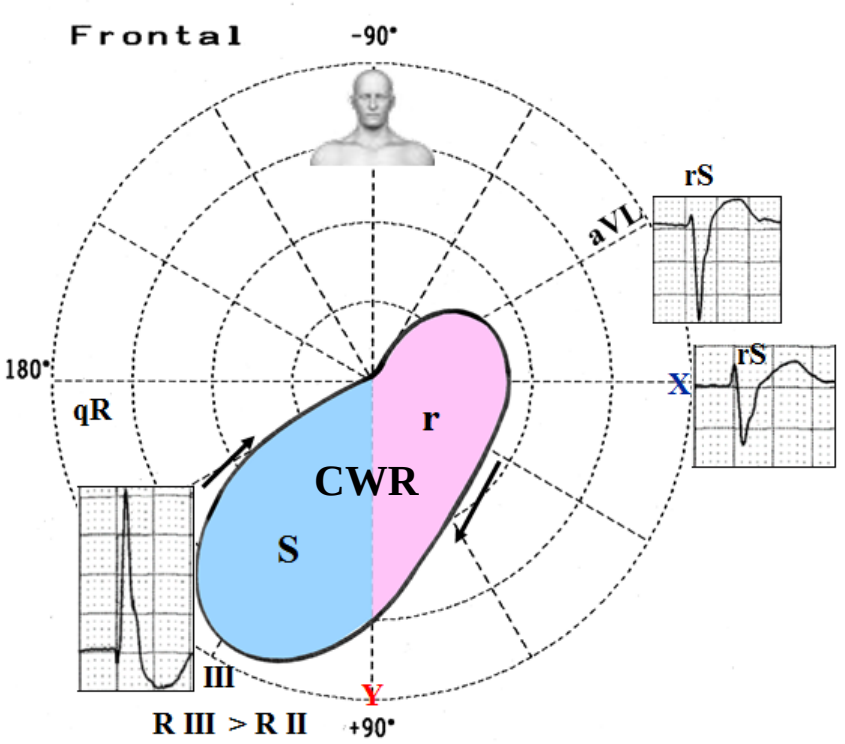
ms 640 609 664 640 648
V V
Y Channel
09:18:45

Typical LPFB pattern of QRS loop in the frontal plane





Characterization of QRS loop in the frontal plane: Vector of initial 20 ms heading above and to the left; efferent limb to the left; clockwise rotation (CWR); greater area of QRS loop located in the right inferior quadrant; maximal vector heading below and to the right near +110º (from +80º to +140º); QRS loop of "broad" aspect ("fat" loop); afferent limb located in the right inferior quadrant. Typical QRS loop in the frontal plane that explains the rS pattern in I and aVL and qR pattern in III with notch in the descending limb of the R wave and R wave in III > R in II (middle-final notch).

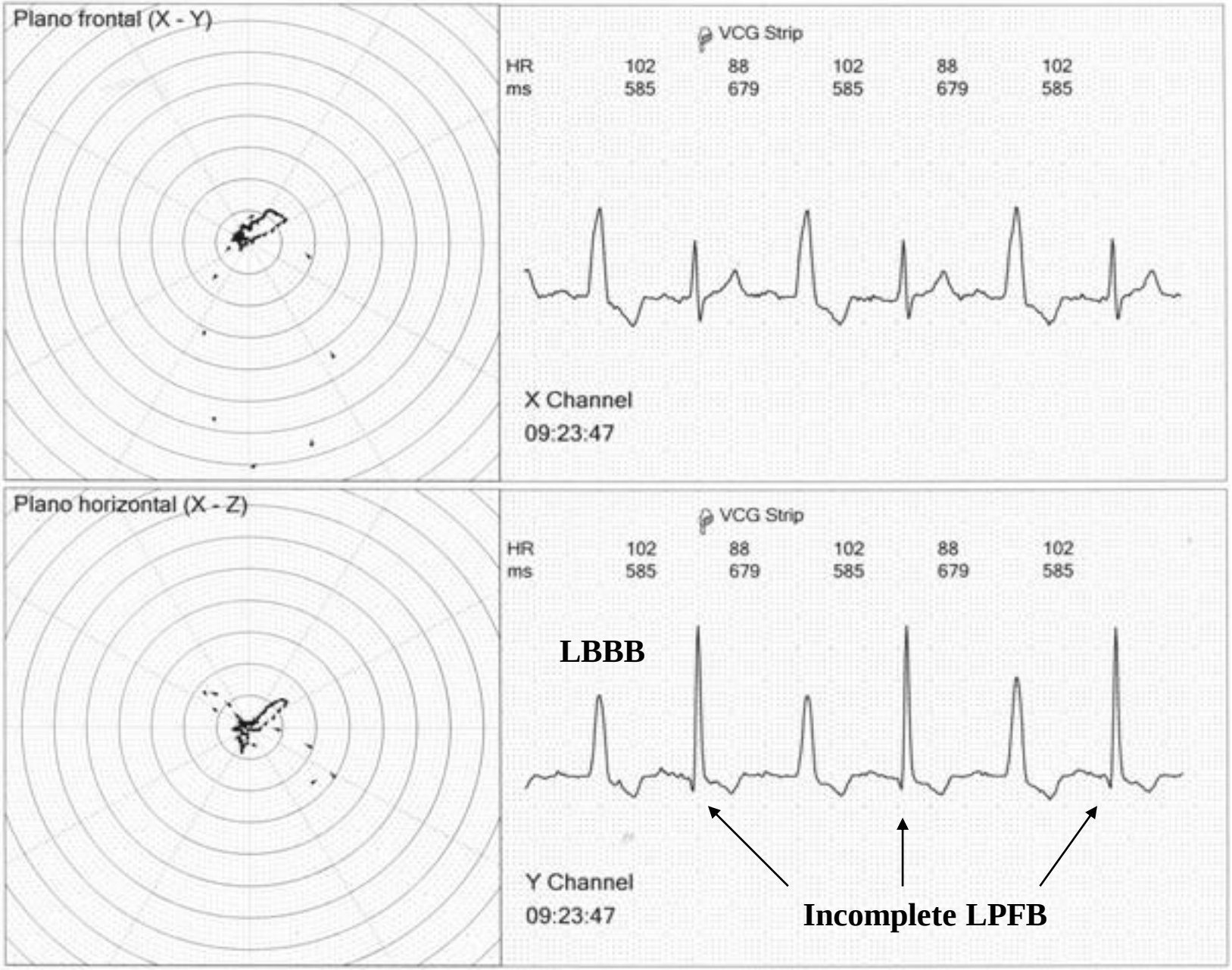
VCG criteria for LPFB (Brohet 1977)

Frontal Plane:

- Vector of initial 10 to 20 ms heading above and to the left (near -45°) with possible delay (initial 10 to 25 ms). If associated to inferior infarction, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms) (Castellanos 1972).
- Broad QRS loop, with clockwise rotation. Cooksey, Dunn and Massie said that occasionally, it may be in "eight" with a counterclockwise terminal portion (10%).
- Maximal vector near +110º (+80º to +140º)
- Almost all the loop is located below the X line (0 to ±1800) in the inferior quadrants
- 20% of the loop located in the right inferior quadrant. If there is association to CRBBB, 40% or more
- Afferent limb heading below and slightly to the left, and the efferent one to the right.
- Middle-terminal portion of the QRS loop (vector of 60 ms to 100 ms) with delay. It may possibly reach the right superior quadrant
- QRS loop duration up to 110 ms if in isolation. In association to Complete RBBB > 120 ms
- Normal ST-T vectors in isolated LPFB: T loop with clockwise rotation, heading below and to the left. If in association to Complete RBBB: alteration secondary to ventricular repolarization.

Horizontal Plane:

- QRS loop very similar to RVH of type C;
- QRS loop of counterclockwise rotation. It is admitted that the rotation could be in "eight";
- Vector of initial 10 to 20 ms heading to the front and the right or left;
- Greater area of QRS loop located in the left posterior quadrant;
- Maximal vector of QRS around -60^0 to -110^0;
- Final portions with delay (60 ms to 100 ms) and located in the right posterior quadrant;
- 20% or more of the area of the QRS loop located in the right posterior quadrant;
- T loop to the front and the left ($+60^0$) and clockwise rotation.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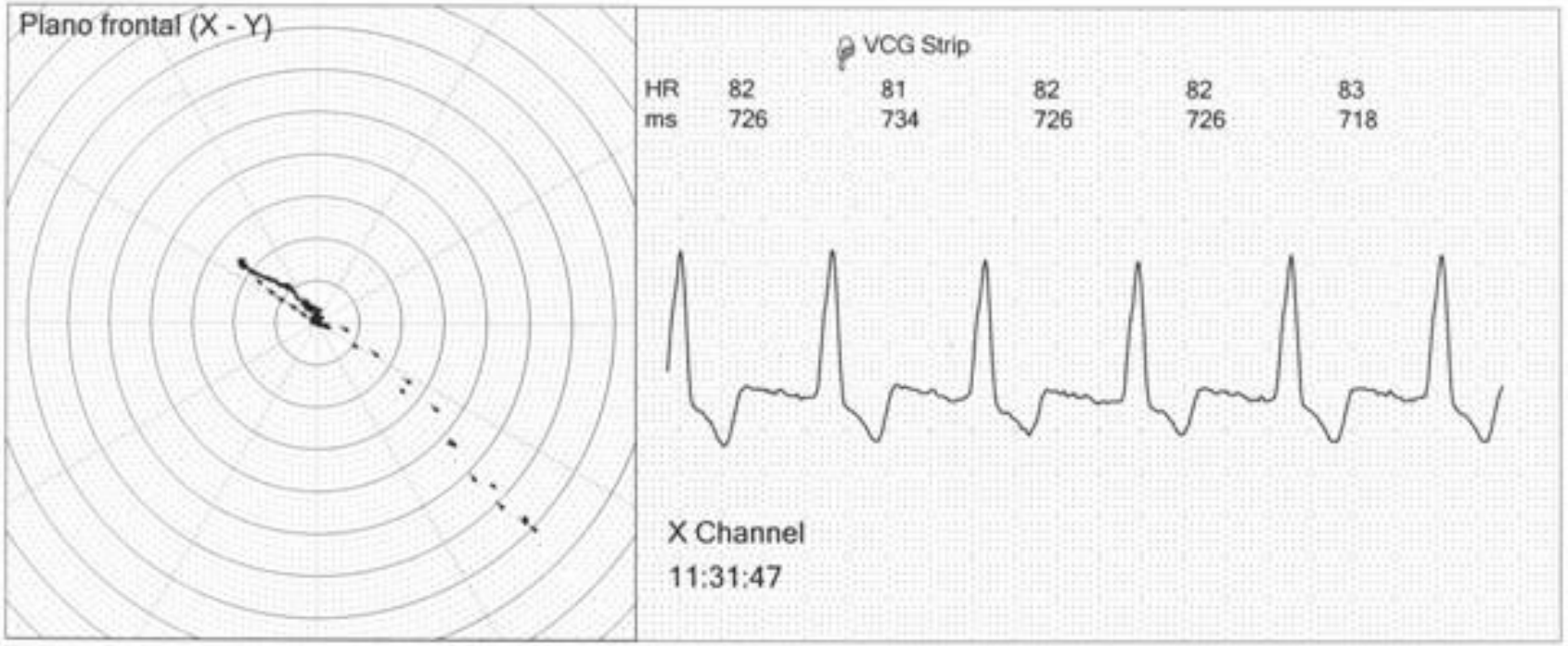
VCG Strip
HR 102 88 102 88 102
ms 585 679 585 679 585
X Channel
09:23:47
Plano horizontal (X - Z)
VCG Strip
HR 102 88 102 88 102
ms 585 679 585 679 585
LBBB
Y Channel
09:23:47
Incomplete LPFB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VCG Strip
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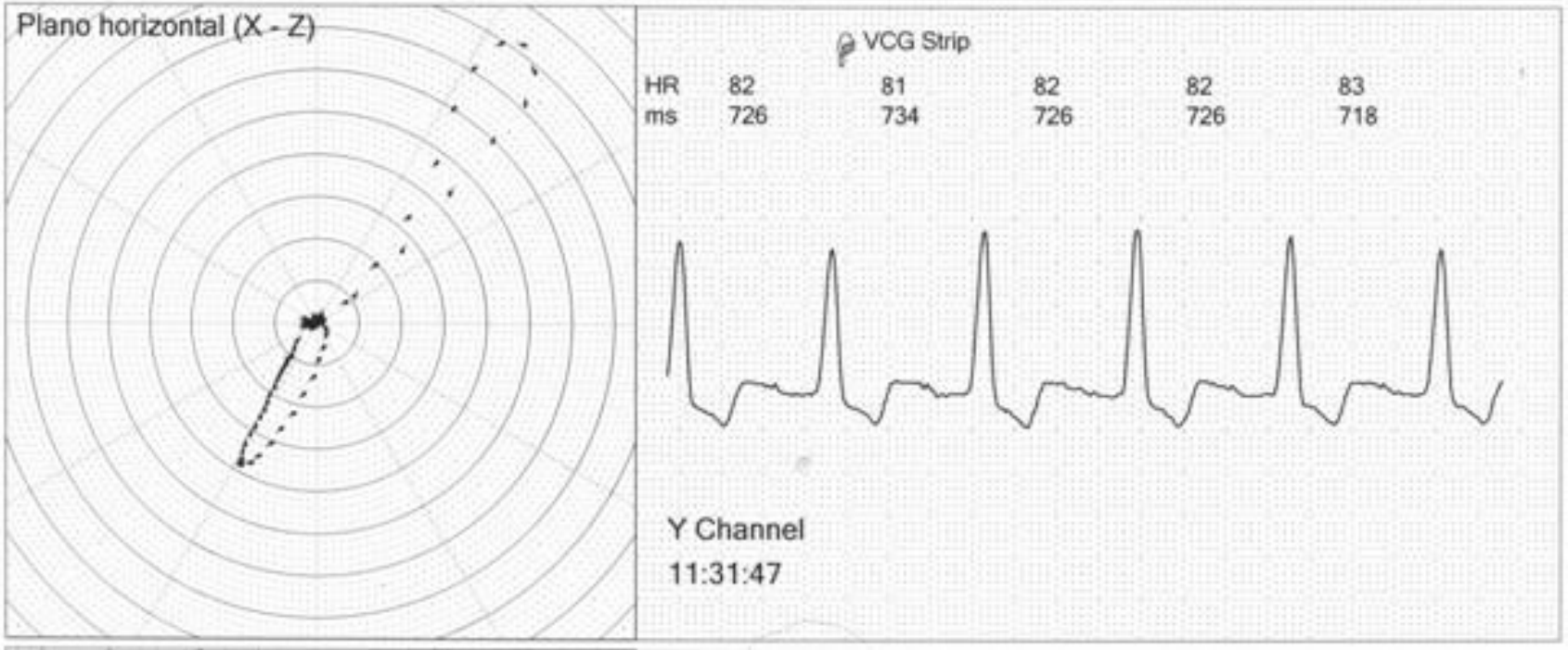
X Channel
11:31:47


Plano horizontal (X - Z)
VCG Strip
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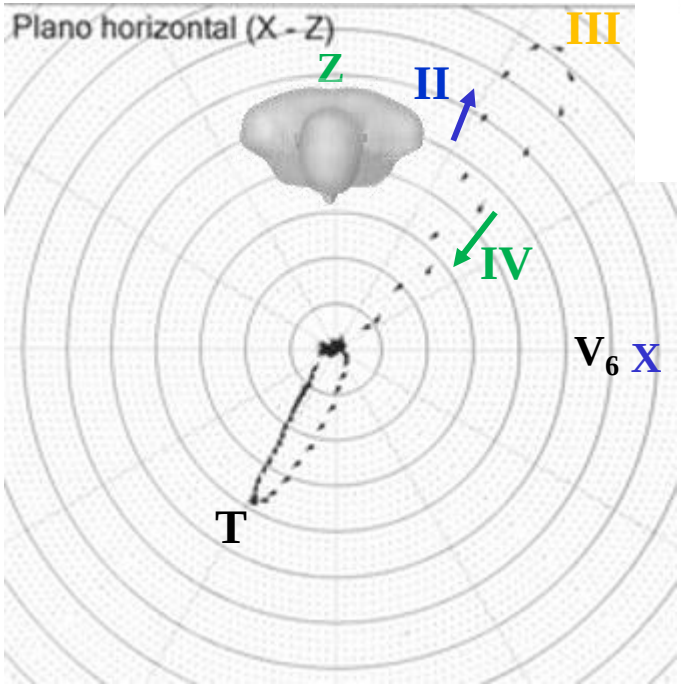
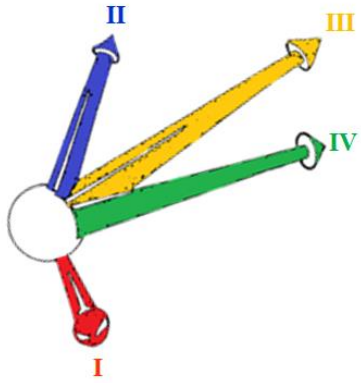
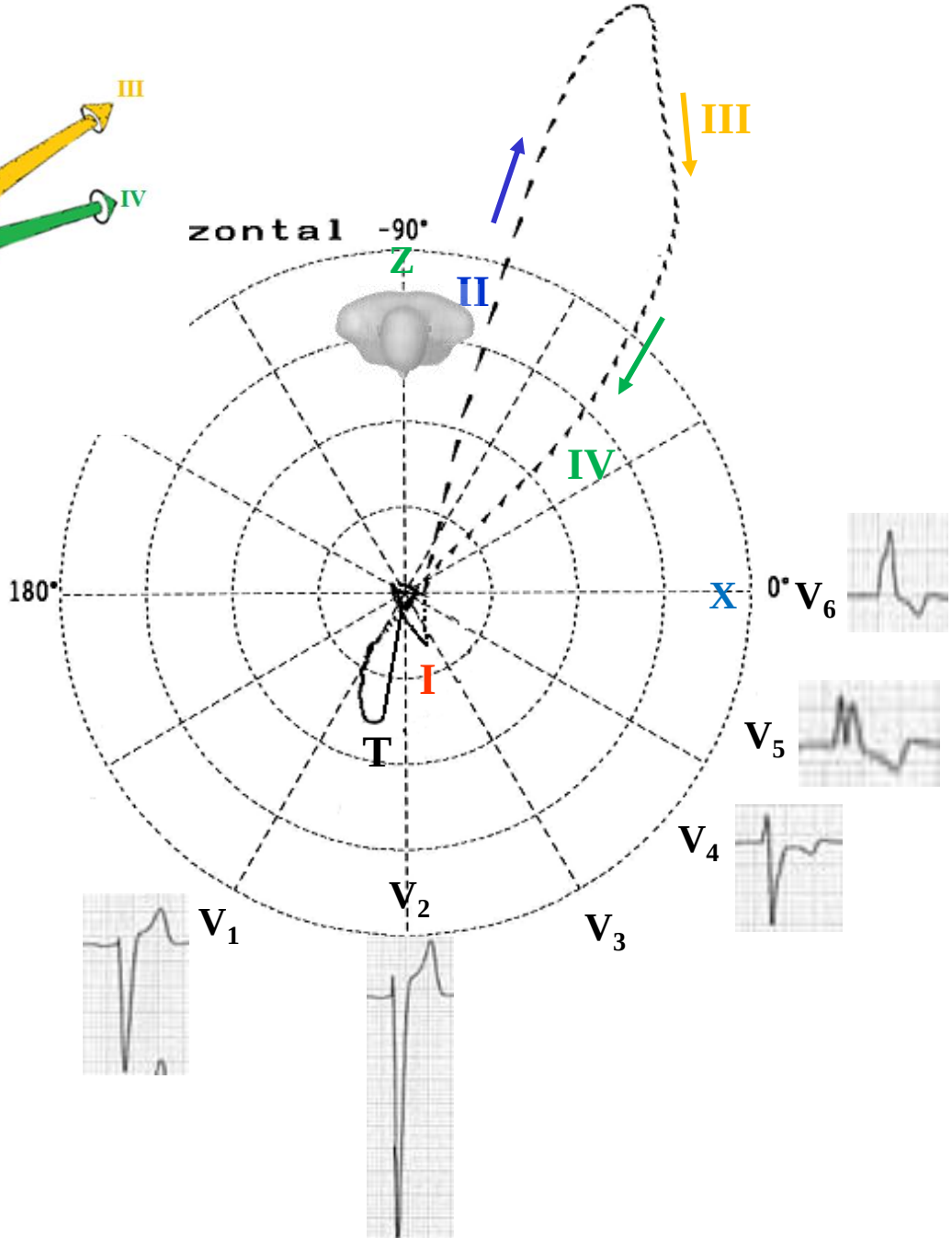
ms 726 734 726 726 718
Y Channel
11:31:47

LBBB pattern in the horizontal plane

The present case







Bundle branch block, unexplained syncope and abnormal EPS

Electrophysiological assessment includes measurement of the His-ventricular (HV) interval at baseline, with stress by incremental atrial pacing and with pharmacological provocation (ajmaline, procainamide or disopyramide). The prognostic value of the HV interval was prospectively studied by Scheinman *et al* (Scheinman 1982); the progression rate to AV block at 4 years was ≤4% in patients with HV interval <70 ms, 12% in those with HV interval of 70–100 ms and 24% in those with HV interval >100 ms. The development of intra- or infra-His block at incremental atrial pacing is highly predictive of impending AV block, but is rarely observed and has low sensitivity. Gronda *et al.* (Gronda 1984) on 131 patients, HV prolongation of >10 ms was observed in 6% and induction of second degree AV block in 5% of cases. Complete AV block developed in 40% of these patients during a mean follow-up of 42 months. In five studies evaluating the diagnostic value of pharmacological stress testing for a total of 333 patients (Bergfeldt 1994), high-degree AV block was induced in 50 (15%) of the patients. During the follow-up of 24–63 months, 68% (range 43–100) of these patients developed spontaneous AV block. By combining the above mentioned parts of the electrophysiological protocol, a positive EPS yielded a positive predictive value as high as ≥80% to identify the patients who will develop AV block (Bergfeldt 1994); this finding has been indirectly confirmed by Moya et al (Moya 2011), that showed a significant reduction in syncopal recurrences in patients with positive EPS treated with PM, compared with a control group of untreated patients with negative EPS. With respect to ESC Guidelines on Syncope, these results justify a recommendation upgrade from class IIa to class I. Thus, in patients with unexplained syncope and bifascicular block, EPS is highly sensitive in identifying patients with intermittent or impending high-degree AV block, though a negative electrophysiological investigation cannot rule out intermittent/paroxysmal AV block as the cause of syncope. Indeed, in patients with negative EPS, intermittent or stable AV block was still documented by ILR in about 50% of cases.

- *Even if the quality of evidence is moderate, there is a strong consensus that patients with positive EPS benefit from pacing therapy. However, the benefit should be weighed against the risk and cost of an invasive procedure.*

Alternating bundle branch block

Alternating BBB (also known as bilateral BBB). Block in the His Purkinje system, rather than block in the AV node or bundle of His, is the most common cause of complete AV block. There are no adequate criteria for determining the precise location of the site of complete heart block from the surface ECG alone. However, it is often possible to diagnose bilateral BBB electrocardiographically prior to the development of complete heart block or during periods of AV conduction returns.

1. Definitive diagnosis of bilateral BBB can be made if the pattern of right and left BBB, accompanied by changes in the PR interval, occur alternately or intermittently in the same patient.
2. If the patterns of RBBB and LBBB appear at different times in the same patient but the PR interval remains constant, the diagnosis of bilateral BBB is reasonably secure.
3. The combination of first-and-second degree AV block with BBB may represent bilateral BBB (i.e., complete block in one bundle branch with incomplete block in the contralateral bundle branch, however when BBB is accompanied by incomplete AV block, the later may be sited in the AV node, the bundle of His, or the other bundle branch. The exact location of the block can only be determined by the use of the His bundle recordings. Thus, the finding of BBB together with incomplete AV block in surface leads can only suggest the possibility or probability of bilateral BBB.

The term trifascicular block refers to the combination of RBBB with intermittent LAFB and LPFB. Trifascicular block is a possibility only when RBBB is associated with either LAFB or LPFB and incomplete AV block (because the AV block may be sited in the AV node, the bundle of His or the left bundle proximally, rather than in the remaining fascicle. There 8 possible trifascicular blocks described by Rosenbaum school.

refers to situations in which clear ECG evidence for block in all three fascicles is manifested on successive ECGs. Examples are RBBB and LBBB on successive ECGs or RBBB with associated LAFB on one ECG and associated LPFB on another ECG. Patients with ECG documentation of alternating BBB are rare. There is general consensus—even if based on anecdotal cases—that these patients progress rapidly toward AV block. Therefore a PM is usually implanted as soon as the alternating BBB is detected, even in the absence of a history of syncope. *Even if the quality of evidence is modest, there is a strong consensus that patients with alternating BBB will benefit from cardiac pacing.*

Bundle branch block, unexplained syncope and non-diagnostic investigations

The ILR experience (Moya 2011) showed that only about half of patients with unexplained syncope and BBB had a documentation of AV block during the period of observation. In a randomized, single-blinded study (Santini 2013), 51 patients with bifascicular block assigned to active DDD 60 bpm pacing were compared with 49 patients with bifascicular block assigned to inactive pacing (DDI 30 bpm). At 2 years, syncope or pre-syncope recurred in 45% of patients in the control group vs. 25% of patients in the treatment group. Overall, a bradycardia was documented in only 14 patients (10 symptomatic AVB, 2 brady-tachy, 1 sinus bradycardia, and 1 permanent AF with slow ventricular response), accounting for an overall incidence of 7.4% per year. Albeit the study showed that cardiac pacing was able to achieve a significant reduction in symptoms, only one out of five patients actually had a benefit and symptoms persisted in a quarter of them.

Thus the decision to implant a PM is determined by an individual risk–benefit evaluation. There are subsets of patients who might receive a favorable cost-effective benefit from this strategy; for example, old patients with unpredictable (no- or very short prodromes) and recurrent syncope that expose them to high risk of traumatic recurrences.

- *The evidence of efficacy of empirical pacing strategy is weak and the estimate of benefit uncertain.*

Indication for cardiac pacing in patients with BBB

Recommendations	Class [a]	Level [b]	Ref. [c]
1) BBB, unexplained syncope and abnormal EPS. Pacing is indicated in patients with syncope, BBB and positive EPS defined as HV interval of ≥70 ms, or second- or third-degree His-Purkinje block demonstrated during incremental atrial pacing or with pharmacological challenge.	I	B	Moya 2011; Twidale 1988
2) Alternating BBB. Pacing is indicated in patients with alternating BBB with or without symptoms.	I	C	-
3) BBB, unexplained syncope non diagnostic investigations. Pacing may be considered in selected patients with unexplained syncope and BBB.	IIb	B	Santini 2013
4) Asymptomatic BBB. Pacing is not indicated for BBB in asymptomatic patients.	III	B	Scheinman 1982; McAnulty 1982; Peters 1979

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